Sleeping Sickness Wakes Again

ROBERT J. BYRNE

Sleeping sickness of horses was, as recently as the 1930's, the most devastating livestock disease in the United States. Horses died by the hundreds of thousands in the Midwestern United States, in California, and along the Atlantic seaboard at a period which coincided with the beginning of the sharp decline in the use of horses on the farm. Losses were so severe in some areas that the inevitable process of farm mechanization, particularly the use of tractors, was accelerated.

Today, the owners of horses, ponies, mules, and burros can take steps to protect their animals from this continuing disease threat.

As used in this chapter, sleeping sickness refers to equine encephalitis, a virus disease of American horses. It should not be confused with African sleeping sickness of human beings which is caused by a blood parasite and spread by bites of tsetse flies.

While severe outbreaks of equine encephalitis had been observed during early periods of American history, the most significant milestones weren't reached until the 1930's in determining its cause, in witnessing its most devastating effects, and in developing control methods.

Credit for recognizing equine encephalitis as a virus disease, and one that is transmitted to horses by mosquitoes, belongs to veterinarians, physicians, and entomologists serving in the U.S. Department of Agriculture

and various biomedical research institutions. Among those who figured prominently were K. F. Meyer, formerly of the University of California and presently Director Emeritus of the Hooper Foundation. USDA veterinarians who contributed to early research on equine encephalitis included H. W. Schoening, M. S. Shahan, and L. T. Giltner.

Investigators soon found that equine encephalitis was due to two different viruses, both producing a similar disease. They were designated as the eastern and western strains of virus. The western virus affects horses generally west of the Appalachians. The eastern attacks horses along the Atlantic seaboard and in the Gulf States. Subsequently, it was found that the two viruses and the disease they produce overlap considerably in their geographic distribution.

Another type of equine encephalitis, affecting horses in South and Central America, is Venezuelan equine encephalitis. However, this disease has not been recognized in horses in the United States.

Meyer was also the first to speculate that the virus causing equine sleeping

sickness might also cause encephalitis in human beings. His theories proved out, as it was soon established that

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ROBERT J. BYRNE is Chief, Research Reference Reagents Branch, National Institute of Allergy and Infectious Diseases, National Institutes of Health, Bethesda, Md. both man and horses are victims of the same disease resulting from exposure to a common source of infection which is found in nature.

Responding to the devastating effect of virus equine encephalitis on American horses in the early 1930's, USDA scientists mounted an intensive research program to understand the disease's nature. They also needed to get this information to veterinarians and animal disease regulatory officials and to develop a control program. Fortunately, the program was highly successful. A satisfactory vaccine emerged by the end of the 1930's.

Pioneer work in the study of vaccines for horses was done by USDA. Development of the most practical vaccine resulted from later discoveries that equine encephalitis virus multiplies readily in developing chick embryos. From these embryos a virus-rich material is obtained which can be made into a potent, safe vaccine.

This finding resulted in commercial production of vaccines which are in present-day use. The vaccines contain both eastern and western strains of virus, since one strain does not protect against the other.

Despite these advances, however, the disease is still a threat to the health of animals and man. To explain this paradox, we need to examine the nature of the virus, the type of disease that it produces, and how it survives in nature and spreads to its victims.

Equine encephalitis is caused by a virus considerably more lethal than viruses causing the common cold, chickenpox, or measles. Like other viruses, it is so small it can't be seen under the ordinary microscope. The virus multiplies in the cells of intact living animals or insects, although it can also be propagated in living cells grown in tissue cultures in the laboratory. As with most other viruses, the equine encephalitis virus is not damaged or killed by penicillin or other antibiotics, so the disease in animals cannot be cured or prevented by using antibiotics. However, chemical agents commonly used as disinfectants do kill the virus and are used in laboratory procedures and in disinfecting.

In the laboratory, the virus can infect and cause disease and death in a variety of experimental animals including mice, guinea pigs, and rabbits. Certain birds, and in particular English sparrows and pheasants, are highly susceptible to the virus and die within days following exposure.

Newly hatched (baby) chicks are highly susceptible, but older chickens are resistant. The virus attacks primarily the brain and so is much more lethal when injected directly into the brain of an animal than, for example, when injected into the muscle or under the skin. This is even true with horses. Only a fraction of horses injected under or into the skin with the virus, or bitten by infected mosquitoes, develop the disease and die.

The virus strains causing western and eastern forms of sleeping sickness are in the family of arboviruses—not to be confused with tree viruses. Arbovirus is a shortened form of arthropodborne animal virus. Arthropods are members of the animal kingdom having an external skeleton and jointed body and legs. They include mosquitoes, ticks, and mites.

Equine encephalitis virus strains are intimately linked to arthropods since they can multiply in mosquitoes which have obtained a virus-containing blood meal from an infected bird or animal. The virus then migrates to the salivary glands of the mosquito where it again multiplies. Thus this mosquito becomes capable of spreading infection, biting, for the rest of its life. The mosquito spreads infection without being adversely affected itself. The relationship of yellow fever, another arbovirus, and the Aedes aegypti mosquito was worked out at the turn of the century by U.S. Army scientists led by Maj. Walter Reed.

Since infected human beings and horses don't have enough virus in their blood to infect mosquitoes, considerable research was needed to determine how mosquitoes become infected in nature. Results of this research revealed the complexity of the equine

encephalitis threat.

It was found that mosquitoes picked up the infection from a variety of wild birds and rodents, some of which reside in almost inaccessible swamps and forests. In these remote areas, the virus can be maintained in a bird-mosquito cycle without doing any apparent harm to domestic animals or man. Because of this, the virus almost defies elimination from the environment.

Under certain circumstances, such as heavy rains and a resultant increase in mosquitoes, the virus breaks loose from its natural surroundings and spreads to birds and other mosquitoes in closer contact with man. The natural cycles for western and eastern forms are not identical nor are the specific mosquitoes that cause their spread. But the basic involvement of wildlife reservoirs presents the same problem in controlling the disease.

A localized outbreak of eastern equine encephalitis which I observed

in 1960 will illustrate a number of factors influencing the number and distribution of cases of disease. In mid-August of that year, a few cases of encephalitis in horses in tidewater Maryland were reported and subsequently confirmed by laboratory tests. The disease continued to occur in Maryland horses and ponies until late September. The population of mosquitoes during this period was quite high. Rainfall was above average during August. Hurricane Donna left very heavy rain along the coast on September 12. No cases of the disease were observed in vaccinated animals.

After several weeks with no new cases being reported, the disease broke out again in the pony herds on Chincoteague and Assateague Islands in Virginia. Most of the deaths were among foals born in the spring of 1960. Many of these young animals had been held past the regular "ponypenning" sale and were used in the filming, during July and August, of

Pony, right, starred as young Misty in the movie "Misty" and died afterwards from equine encephalitis. Many ponies in the film were from wild herds on Virginia islands.



the motion picture, "Misty." A pony which had portrayed young Misty in the film was one of the first to die from the disease in October. At least 30 ponies, none of which had been vaccinated, died on Assateague and on Chincoteague from October 10 to 20.

This outbreak of equine encephalitis had two phases. The first phase represented scattered deaths in tidewater Maryland among horses exposed to the high concentration of mosquitoes resulting from above average rainfall and continued warm weather through much of September. The second phase, the outbreak on Chincoteague and Assateague, followed by 2 to 3 weeks a deluge of rain, from Hurricane Donna. This, plus continued warm weather, resulted in a new wave of mosquitoes—many of which had obtained the infecting virus. The crop of young, susceptible ponies on the coastal islands of Virginia was thus vulnerable to this situation.

Horses infected with encephalitis virus from the bite of a disease-carrying mosquito may react in a number of ways. As previously stated, a significant number show no ill effects—in fact such horses probably become immune to the infecting virus for life.

Other infected horses develop only a slight fever and recover to normal in a few days.

The most severe form is encephalitis or inflammation of the brain. In this form, the disease is commonly recognized, and more than half the horses which develop brain inflammation die.

Early indications of disease which would attract the owner's attention are signs of depression, lack of appetite, and drowsiness—hence the name sleeping sickness. Animals frequently grind their teeth and stagger slightly when moving. While the disease progresses, animals become reluctant to move and are inclined to lean against walls, fences, or other structures. In the final stages, affected horses stagger blindly and finally fall. When down, they continue an aimless, running motion until they die or are killed.

Sometimes an animal with equine

encephalitis doesn't reach the severe stage and may recover completely and probably be immune for life. Those which are most severely affected, and yet survive, are likely to be left with permanent brain damage—the so-called dummy horses. Little can be done to treat sleeping sickness of horses once recognizable signs occur.

However, a veterinarian should be called when the disease is suspected. First of all, the veterinarian may determine that the suspect horse has a disease other than equine encephalitis, and this may be a disease that can be treated successfully. Second, it is important that veterinarians obtain confirmation of equine encephalitis cases in order to inform the animal disease control authorities.

Control measures, including vaccination and mosquito suppression, can be launched or intensified and the lives of other animals saved.

Since there are no specific treatments of any value for equine encephalitis, great emphasis should be placed on preventing the disease. In most U.S. areas, the owners of horses, ponies, mules, and burros should have their animals vaccinated. This is particularly desirable in the areas where the disease is known to occur. Vaccination also is strongly recommended for any horses moved from place to place for shows and races.

Because of the overlap in distribution of eastern and western types of equine encephalitis, bivalent vaccines are recommended, containing a protection against both types.

Horses and other equine animals in risk areas should be vaccinated annually several months in advance of the usual time of the disease's occurrence. August and September are the months equine encephalitis is most frequently observed, so vaccination should take place toward the end of spring. If vaccination has not been carried out due to oversight, it is still worthwhile, even in the face of an outbreak.

A necessary part of prevention is mosquito control, which is primarily

in the hands of State or local government. Mosquito control measures are most beneficial if applied early in an outbreak of equine encephalitis and when directed at the particular mosquitoes implicated in its spread. Mosquito control efforts are highly effective in preventing the spread of yellow fever which is carried by the Aedes aegypti mosquito, an urban area dweller. But the complex natural history of eastern and western types of equine encephalitis makes mosquito control measures against this virus more difficult. Continued research may solve some of the problems.

The individual horse owner can apply mosquito control measures in horse stables and transportation vans. Recommendations for safe, effective sprays can be obtained from county agents and local mosquito control authorities. Bringing pastured animals indoors during the night reduces exposure to mosquitoes that carry and spread the infection.

Although man and horses are both

victims of the same disease, it is not spread from horses or man. Both contract the disease from a common source in nature. If you compare the number of people in the United States with the number of horses and then examine the frequency of the disease in each, it becomes quickly obvious that the disease in man is quite rare. In horses, the attack rate is higher because they are openly exposed to heavy concentrations of mosquitoes in areas where equine encephalitis occurs.

In summary, the health of animals and man have benefited from research on equine encephalitis (sleeping sickness) virus. Effective vaccines have been developed. The mosquito control measures have been strengthened. And improved laboratory techniques have speeded diagnosis of the disease so that quick action can be taken to save lives. More advances will come through the coordinated efforts of physicians, veterinarians, entomologists, microbiologists, and other scientists who make up the research team.

Knock on Any Door: Consumer Research

THEODORE R. CRANE

Household surveys of consumer opinion, studies of reactions to new foods and fibers, all help bridge the gap between the farmer and his ultimate customers. It is a distance that may span hundreds or even thousands of miles, a marketing system that includes a crowd of intermediary handlers and processors.

Mrs. McCann, with her six kids, husband, one dog, and an occasional stray cat, lives in a comfortable rambling house on the edge of the small western town where her husband runs a prospering hardware store.

When she thinks of food—which is

often, with a family her size—she thinks first of food that the entire family will like.

Mrs. Hoffman's home, a modest brick structure, is in a development not far from the shores of Lake Michigan. The house seems a bit crowded,